US EPA Mid-Continent Ecology Division

Research Project Summary

Effects of Dietary Metal Exposure on Aquatic Organisms

Overview

Conventional wisdom in aquatic toxicology is that water is the primary exposure route determining the toxicity of most cationic metals (e.g., Cu, Zn, Ni, Cd, Pb) and metal mixtures to fish and other aquatic organisms. Although exposure to metals via the diet was known to produce some level of bioaccumulation, toxicity from dietary intake did not appear to be a significant pathway in determining toxicity to fish, with the exception of mercury, selenium, and perhaps arsenic. Thus, environmental criteria and other toxicity assessments for Cu, Zn, Ni, Cd, and Pb (hereafter referred to as "metals") have focused on water column toxicity.

Beginning in the mid-1990s a series of dietary toxicity studies was conducted (Woodward et al. 1994, 1995; Farag et al. 1994) that involved feeding young rainbow trout diets prepared from invertebrates collected from metal-contaminated rivers, primarily the Clark Fork River (CFR) in Montana. Results of these studies showed that fish fed a diet of pellets prepared from metal-enriched invertebrates had reduced growth relative to fish fed similar diets prepared from invertebrates from reference areas or less contaminated portions of the CFR. The Clark Fork watershed is enriched with several metals, though copper is generally considered to be the metal of greatest concern. A more recent study from the same laboratory (Farag et al. 1999) reports comparable findings for invertebrates from the Coeur d'Alene watershed in Idaho, where the primary metals of concern are lead, cadmium, and zinc. However, similar feeding studies conducted with laboratory-prepared diets have not indicated substantial effects of dietary exposure to many common metals except at extreme concentrations.

The repercussions of this issue in regulatory programs are large and persistent. The significance of dietary exposure has not only been a major point of contention in the assessment of Clark Fork River, but has also infiltrated debates on a number of regulatory issues, including the adequacy of ambient water quality criteria for metals, the advisability of assessing waterborne metals on the basis of dissolved (rather than total recoverable) metal, and the adequacy of EPA's proposed chemical-specific sediment guidelines (formerly Sediment Quality Criteria) for metals. Dietary exposure to metals is also a major point of debate in the ongoing Endangered Species Act consultation on water quality criteria currently underway among EPA/OW, EPA/ORD, USFWS, and NMFS. None of EPA's environmental criteria currently consider dietary exposure to metals as part of assessing risk. Moreover, the technical debate surrounding the issue is mired in conflicting and insufficient data with no clear resolution of the biological significance of the dietary pathway, much less a way of quantifying the risk for incorporation into environmental regulation (See Mount et al. [1994] and Erickson [2001] for a more extensive discussion of the issues surrounding the dietary exposure issue.)

In our view, the dietary metal exposure pathway is currently lacking in scientific credibility because of an inability to reproduce the effect with experimentally-prepared diets in a controlled

setting. Although diets prepared from field-collected diets have been associated with biological effect in repeated experiments, field-collected diets suffer from potential confounding variables that occur in the field (e.g., different nutritive value between sites). Effects of dietary metal exposure have been demonstrated in some laboratory studies using inorganic metal salts added to diets, but only at exorbitantly high concentrations of metal and often associated with food refusal and/or vomiting. The most effective approach to understanding the toxicity of dietary metals is to simulate the dietary exposure experienced under field conditions using natural diets of live organisms reared under conditions relevant to the exposure of prey organisms in the field. If effects of dietary metal exposure can be induced under these conditions, we will have both firmly established the legitimacy of the exposure pathway and provided a reproducible experimental system under which the mechanism and toxicokinetics of the pathway can be studied. Understanding of the mechanism is critical to both establishing the appropriate dose metrics and resolving the apparent discrepancies in results obtained previously using different experimental systems. If the proposed studies do not show effects from dietary exposure to metals, it cannot be taken as proof that the pathway is not significant (that would involve proving a negative) but will provide EPA risk assessors with support for the current approach of focusing on waterborne exposure as the primary pathway for risk.

Key Products

Journal manuscript on the toxicity of dietary exposure of rainbow trout, fathead minnows, and channel catfish to Cu, Cd, Zn, and Pb.

For further information on this research contact:

Russ Erickson / Dave Mount
erickson.russell@epa.gov
529-5157